EXHIBIT C-2

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RE: Toy Sr., Thomas H. (DOB May 25, 1935)

I, Brent C. Staggs, M.D., am over 18 years of age, being duly sworn, depose and state as follows:

I am a physician and partner with Pathology Laboratories of Arkansas, P.A. I received by medical degree from the University of Arkansas for Medical Sciences in 2001 and subsequently completed by residency training at the University of Arkansas for Medical Sciences Department of Pathology from 2001 thru 2007. During that time, I completed my fellowships in hematopathology and gastrointestinal pathology. During this time, I had extensive training in pulmonary pathology and molecular pathology, which I have continued to practice with interest throughout my career. I am board certified in Anatomic Pathology, Clinical Pathology and Hematopathology.

I am currently licensed to practice medicine in Arkansas, Alabama, Mississippi, Oklahoma and Texas. I have been a licensed Medical Review Officer since 2009, with re-certification in 2014. Since 2007 I have practiced with Pathology Laboratories of Arkansas, P.A. As part of my practice and on a daily basis, I examine numerous tissues, including tumor types and lung biopsies. I review molecular analysis of numerous types of pathology specimens, both tissue and blood, including molecular analysis of lung cancer and mesothelioma. I am the medical director for multiple laboratories including the largest clinical laboratory in the state of Arkansas, the Baptist Health Medical Center in Little rock. During my career, I have reviewed hundreds of cases of asbestosis,

asbestos related pleural disease, lung carcinomas and mesotheliomas, both in clinical practice and in the setting of medical legal consultation. I have performed more than a thousand autopsies, which included individuals who suffered from asbestos-related diseases. My qualifications are further described in my Curriculum Vitae (Exhibit 1).

I hold the following opinions to a reasonable degree of medical and scientific certainty based on my personal training, experience, knowledge, and expertise, as well as relevant literature, articles, studies, documents, and medical and scientific principles. The methodology and bases for these opinions are generally accepted in the medical and scientific community. While I do not agree with every position or statement of each document or reference cited, the materials and information discussed below are appropriate and sufficient to form a reliable basis for my opinions. These are the types of foundational materials that I, as well as other medical doctors rely upon in our daily and standard practice of medicine to formulate opinions outside the medical legal consulting context. This affidavit may be submitted in litigation to accompany case-specific medical reports and/or for general disclosures as a basis for my opinions and methodology. This affidavit does not, nor is it intended to, state all of my opinions or all of the supporting materials on the related subject matter.

Principles of Asbestos Related Diseases

1. Asbestos is a term used to describe two families of naturally-occurring fibrous minerals, serpentine (chrysotile) and amphibole (actinolite, amosite, anthophyllite, crocidolite and tremolite). Asbestos was mined for commercial purposes in Canada, South Africa, Italy, Australia, Rhodesia, Russia, United States, and other countries, starting in the late 1800's. The list of commercial uses for asbestos grew so long that by 1972, the uses were summarized into broad areas consisting of construction, floor tile, friction materials, paper asphalt felts, packing and gaskets, insulation, textiles, and other. Selikoff, Irving J., and D.H.K. Lee. Asbestos and Disease. Academic Press, NY, 1978.

2. Asbestos disease in humans was described in reports in many countries as early as late 1890's through the 1920's. In 1930 Merewether and Price

published an official government investigative report of conditions and health, problems, including asbestosis, related to the asbestos industry in England. Merewether then published a lengthy report of his findings in the United States in the same year. Merewether, E.R.A. The Occurrence of Pulmonary Fibrosis and Other Pulmonary Affections in Asbestos Workers. J Indust. Hyg. 12:198-222, 239-257 (1930). Castleman, B., Asbestos Medical and Legal Aspects, 5th Ed., 2004.

3. Asbestos was causally linked to cancer, starting with case reports, in the 1930's and 1940's. Lynch, K. M. and W. Atmar Smith. Pulmonary asbestosis III: Carcinoma of Lung in Asbestos-Silicosis. Am. J. Cancer. 24:56 (1935). In 1949, additional references implicated asbestos as a cause of cancer. Asbestosis and Cancer of the Lung, The Journal of the American Medical Association (JAMA), Vol. 140, No. 15, pp. 1219-20 (1949); Smith, L., Pneumoconiosis and Lung Cancer, Compensation Medicine, Nov (1949); Conklin, G., Cancer and Environment, Scientific American, Vol. 180, No. 1, Jan (1949). By the 1950's, asbestos was well-known and accepted as a cause of cancer. Doll, R. Mortality from Lung Cancer in Asbestos Workers. Br. J. Ind. Med. 12 (2):81-86 (1955). In addition, mesothelioma was reportedly caused by asbestos in a number of sources in the 1950's. The fact that asbestos caused mesothelioma was firmly established during the 1960's. For discussion, see Selikoff, Irving J., and D.H.K. Lee. Asbestos and Disease. Academic Press, NY, 1978 and Castleman, B., Asbestos Medical and Legal Aspects, 5th Ed., 2005. Chapter 2 - Cancer.

4. Asbestos is a known and complete carcinogen, so it both initiates and promotes cancer. It has been demonstrated for decades through scientific study that the inhalation of asbestos fibers of all types causes mesothelioma, lung cancer, as well as cancer of other sites. In addition to cancer, asbestos causes non-malignant diseases including asbestosis, pleural fibrosis, and pleural plaques. The ability for all types of asbestos (amphibole, chrysotile, as well as non-commercial asbestiform mineral fibers) to cause cancers and non-malignant disease is overwhelmingly accepted and agreed to by the medical and scientific community. This scientific consensus is reflected many sources and

publications, and the references contained therein, upon which I rely, including:

- Toxicological Profile for Asbestos. U.S. Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Sept 2001.
- American Thoracic Society. Diagnosis and initial management of nonmalignant disease related to asbestos. Am J Respir Crit Care Med 2004; 170:691-715.
- IARC. Asbestors: Monograph on the Evaluation of Carcinogenic Risk to Man. Lyon: International Agency for Research on Cancer (1988). IARC: Monograph on the Evaluation of Carcinogenic Risks to Humans (2012); 100C:219-309.
- Helsinki Consensus Report. Asbestos, asbestosis and cancer: The Helsinki criteria for diagnosis and attribution. Scand J Work Environ Health 1997. Helsinki Consensus Report. Asbestos, asbestosis and cancer: The Helsinki criteria for diagnosis and attribution. Scand J Work Environ Health 2014.
- Henderson DW, Rodelsperger K, Woitowitz H-W, Leigh J. After Helsinki: A multidisciplinary review of the relationship between asbestos exposure and lung cancer, with emphasis on studies published during 1997-2004. Pathology 2004; 36:517-550.
- Welch et al., Asbestos exposure causes mesothelioma, but not this asbestos exposure: An amicus brief to the Michigan Supreme Court. Int. J. Occup. Environ. Health 13:318-327 (2007).
- American Conference of Governmental Industrial Hygienists. Asbestos: TLV ® Chemical Substances 7th Edition, ACGIH Report No.: Publication #7DOC-040 (2001).

- National Toxicology Program. Report on Carcinogens, Eleventh Edition. U.S. Dept. of Health and Human Services, Public Health Service (2005).
- Occupational Safety and Health Administration. Occupational exposure to asbestos; final rule. Federal Register, 59:40964-t 162 (1994).
- Environmental Protection Agency. Airborne Asbestos Health Assessment Update. Springfield VERMIFORM APPENDIX: NTIS; Report No.: EPA/600/8-84/003F (June 1986).
- World Health Organization. Chrysotile Asbestos. 2014; World Health Organization. Elimination of asbestos related diseases. Ref Type: Generic (2006).
- Collegium Ramazzini, The Case for a Global Ban on Asbestos. Environ Health Perspectives 118:897-901 (2010). Collegium Ramazzini, Comments on the 2014 Helsinki Consensus Report on Asbestos. Oct. 14, 2015. Collegium Ramazzini, Comments on the Causation of Malignant Mesothelioma, Oct. 14, 2015.
- World Trade Organization European Communities - Measures Affecting Asbestos and Asbestos - Containing Products. Report No.: WT/DS135/R (2000).
- Brody, A.R. "Asbestos". In: Comprehensive Toxicology, Pergamon Press, Vol. 8- Toxicol. of the Resp. system (Ed. R. Roth), pp. 393-413, 1997.
- Lemen RA. Dement JM, Wagoner JK. Epidemiology of asbestos-related diseases. Environ Health Perspect 1980; 34:1-11.
- Lemen RA. Chapter 5 - Epidemiology of Asbestos-related Diseases and the Knowledge that Led to What is Known Today. In: Dodson RF, Hammar SP (Eds.), Asbestos: Risk Assessment, Epidemiology, and Health Effects, 2nd ed. CRC/Taylor & Francis, 2011:131-267.

- Stayner L, Welch LS, Lemen R. The worldwide pandemic of asbestos-related diseases. Annu Reveals Public Health 2013; 34:205-216.
- Markowitz, S., Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues, Seminars in Respiratory and Critical Care Medicine, Vol 36, No 3/2015.
- Kanarek, MS. Mesothelioma from Chrysotile Asbestos: Update. Ann Epidemiol 2011;21(9) 688-697.
- Dodson RF, Hammar SP, eds. Asbestos: Risk Assessment, Epidemiology, and Health Effects, 2nd Ed. Boca Raton: CRC Press/Taylor & Francis Group, 2011. Note: 1st edition published in 2006.
- Dail and Hammar's Pulmonary Pathology, 3rd Ed. Vols I and II. New York: springer, 2008.
- Kumar V, Abbas AB, Fausto N, Aster JC (eds). Robbins and Cotran Pathologic Basis of Disease, 8th Ed. Philadelphia, PA: Saunders/Elsevier, 2010.
- Suzuki, et al., Asbestos tissue burden study on human malignant mesothelioma, Ind. Health, 39:150-160(2001); Suzuki, et al., Asbestos Fibers Contributing to the Induction of Human Malignant Mesothelioma. Ann. N.Y. Acad. Sci. 982: 160-176 (2002); Suzuki, et al., Short, thin asbestos fibers contribute to the development of human malignant mesothelioma; pathological evidence. Int. J. Hyg. Environ Health 208: 201-210 (2006).
- Gao Z, et al., Asbestos Textile Production Linked to Malignant Peritoneal and Pleural Mesothelioma in women: Analysis of 28 Cases in Southeast China, American Journal of Ind. Med. 58:1040-1049 (2015).

5. Asbestos shows a linear dose-response relationship with regard to the risk of developing mesothelioma, lung cancer and non-malignant asbestos

diseases. It is demonstrated in multiple studies that the more asbestos a person is exposed to over time, the higher the risk of developing an asbestos related disease. See above references; see also Pitot, Fundamentals of Oncology; 4th Ed., Marcel Dekker, NY, 2002. This risk is related to the cumulative dose of asbestos inhaled over time, and all doses (small and large) of asbestos contribute to the cumulative dose. When more asbestos fibers are inhaled, more asbestos fibers overcome the body's defense mechanisms, which increases the risk and causes an asbestos disease(s) in some individuals.

6. When asbestos is inhaled, a portion of asbestos fibers are retained in the lungs and others are translocated to other sites where disease develops. Miserocchi et al., Translocation pathways for inhaled asbestos fibers, Environ. Health 7:4;1-8 (2008). Mesothelioma occurs when asbestos fibers cause multiple/sufficient genetic errors in mesothelial cells around the lungs (pleura), the abdomen (peritoneal), the heart (pericardium), or the reproductive organs (tunica vaginalis). All types of asbestos induce genetic errors. Kamp, D.W. Asbestos-induced lung diseases: An update. Translational Res. 153: 143-152, 2009. Hei, T.K. et al. Chrysotile is a strong mutagen in mammalian cells. Cancer Res. 52: 6305-9, 1992. Harris, C.C., Lechner, J.G. and Brinkley, B.R., Cellular and Molecular Aspects of Fiber Carcinogenesis. Cold Spring Harbor Lab Press, NY, 1991. It often takes many decades (latency period) for a sufficient number of genetic errors to result in a mesothelioma, as the body has defense mechanisms which destroy defective cells.

7. I am in agreement with the methodology discussed and employed by IARC, ATSDR and others, which includes looking at all available evidence regarding the dangers of asbestos. This weight of evidence approach is consistent with the Bradford Hill Criteria that is often referenced, as well as Dr. Lemen's application. Lemen, R.A., Chrysotile. Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model, Int. J Occup Environ Health, Vol. 10, No. 2, Apr/June 2004.

8. All governmental and authoritative agencies have reviewed appropriate scientific studies and literature and all have determined that there is no safe level

(threshold) of asbestos exposure below which there is no risk of cancer. Asbestos exposure limits allowed by government regulations are not, nor were they intended to be protective of cancer. See referenced sources, including EPA, NIOSH, USPHS, OSHA, and WHO. Rather, these allowances are a compromise between industry feasibility, industry resistance to asbestos regulations, and the protective goals of the agencies. The scientific community knows that exposure to asbestos at the regulatory levels will result in excess cancers.

9. The potency of fiber types to cause mesothelioma and lung cancer is an unsettled issue. Numerous studies have reported potency differences between chrysotile and amphiboles and many theories and explanations exist. Markowitz SB, 2015. The IARC Monograph on asbestos from 2012 reported there is a high degree of uncertainty concerning the accuracy of the relative potency estimates derived from the Hodgson & Darnton and Berman & Crump analyses. Further complicating the issue is mixed-fiber exposure, which regularly occurs in individuals with asbestos caused cancer. See also, Patty's Toxicology, Sixth Ed. Vol 5. Chapter 83. John Wiley & Sons, 2012.

10. Mesothelioma is a rare and fatal cancer, reportedly occurring in approximately 3,000 persons per year in the United States. Although there are likely more cases that are not identified in tumor surveillance programs. The relationship between mesothelioma and exposure to asbestos is so well established in the scientific community, that mesothelioma is considered a "signal tumor" for asbestos exposure. See also, Reference Manual on Scientific Evidence, Federal Judicial Center 1994 and 2000 Editions, p. 351, "There are some diseases that do not occur without exposure to an agent; these are known as signature diseases." See also Shy, Greenberg, Winn, Sentinel Health Events of Environmental Contamination: A Consensus Statement, Nov. 1, 1989; ATSDR, Case Studies in Environmental Medicine: Asbestos Toxicity, Rev. Nov. 2000. Thus if a person has mesothelioma, it signals that they likely had prior asbestos exposure, even if they are not aware of such exposures or do not remember them from many years earlier. It is also possible that upon biopsy or autopsy, no asbestos fibers are

found, yet asbestos fibers were previously present, which caused genetic errors leading to mesothelioma.

11. It has been established that household/take-home and neighborhood exposures cause mesothelioma. See supra para 4, and see also, Wagner, 1960 (Neighborhood); Newhouse, 1965 (Household and Neighborhood); Lieben and Pistawka, 1967 (Household, Neighborhood and Low Dose p.561 patients 1Q - 10Q); Selikoff and Lee, 1978, p 265-275 (Household and Low Dose); Hillerdal, 1999 (Household, Neighborhood and Low Dose); Magnani, 2000 (Household, Neighborhood, and Low Dose); Ferrante, Mirabelli, et al, 2015, (Household, Neighborhood and Low dose).

12. It has been established that lower range and short-term exposures (often characterized as "lose dose") cause mesothelioma. See supra para 4 and para 11 and see also Greenberg, 1974; Chen, W., 1978; Iwatsubo, 1998; Magnani, 2000; Rodelsperger, 2001; Skammeritz, 2011; LaCourt, 2014; Offermans, 2014. and Markowitz, S., 2015.

13. Studies have also shown that lower doses of asbestos exposure increase the risk of lung cancer. Van der Bij S, Koffijberg H, Lenters Vk, et al. Lung Cancer Risk at Low Cumulative Asbestos Exposure-Response Relationship. Cancer Causes Control, 2013; 24(1):1-12. Further, asbestos caused lung cancer may occur without the presence of asbestosis. Although if asbestosis is present, the risk of lung cancer increases even more because if demonstrates a greater exposure to asbestos. Markowitz SB, Levin SM, Miller A, Morabia A. Asbestos, asbestosis, smoking and lung cancer. New findings from the North American insulator cohort. Am J Respir Crit Care Med. 2013; 188(1):90-96. Markowitz SB, 2015. See also, Gustavason P, Nyberg F, Pershagen G, Scheele P, Jakobsson R, Plato N. Low-Dose Exposure to Asbestos and Lung Cancer: Dose-Response Relations and interaction with Smoking in a Population-based Case-Referent Study in Stockholm, Sweden. Am J Epidemiol. 2002; 155(11):1016-1022.

14. The mechanisms by which asbestos causes mesothelioma and lung cancer are now being borne out more clearly with on-going research. Similar to other malignancies, asbestos cancers happen only after a series of numerous

deleterious mutations occur over time. A single mutational event does not "cause" a mesothelioma or lung cancer by chance. Inhaled asbestos fibers are either directly carcinogenic to exposed cells or they induce inflammation and that inflammation causes mutational events in exposed cells. It takes multiple mutations to "drive" the malignant cell population. This mechanistic approach explains how multiple or cumulative exposures contribute to the development of a mesothelioma or lung cancer.

15. In genetically susceptible individuals, a lower level of asbestos exposure will be needed to develop or cause asbestos cancer, as opposed to a less susceptible individual. A given individual's susceptibility to asbestos cancer cannot usually be identified; however, there has been recent interest in this area. Most notably, there have been studies involving mutation of the BAP1 gene locus, which determined that those with BAP1 are predisposed to developing malignant mesothelioma. The BAP1 gene was found to cause dysregulation of inflammation induced by asbestos.

16. It is my opinion that mesothelioma and lung cancer are dose-response diseases that are caused by the cumulative exposures to asbestos that a person received during their lifetime, minus an appropriate latency. By "dose" I mean the amount of asbestos inhaled over a given time period. By "exposure" I do not mean a single fiber, rather I am referring to thousands and millions of asbestos fibers commonly released by asbestos containing products. An exposure or dose is defined by the context of a given case, i.e. exposure related to a premises, employer, specific product etc. By "dose-response" I mean that the more asbestos exposures someone has, the more likely it is that they will have a response to the dose, like development of a cancer. It is the cumulative dose that causes the repeated changes and mutations that result in the cancer. Although all exposures large and small must be considered to create a given individual's cumulative dose, not all parts of the cumulative dose are significant contributors. The issue of significant contributing parts of an individuals overall cumulative dose cannot be defined by a single cutoff number or calculation of exposure because we are all different biologic organisms with different susceptibilities. The issue of significance must be considered by weighing all of the pertinent and available

factors related to that individual's exposures. As a physician, I never deal with a patient who presents with a pre-calculated cumulative dose such as a numeric value (i.e. fiber/cc years). A physician considers exposures to substances based on the history given by the patient. A few of the factors are how closely a patient worked with a product, how often the products was used and in what manner. These are only a few of the factors to consider when assessing the significance of one part of a cumulative dose. This is the same semiquantitaive approach that I and other physicians use to evaluate toxic exposures (not just to asbestos) in my daily practice. This approach is summarized in the 2012 article Assessing specific causation of mesothelioma following exposure to chrysotile asbestos-containing brake dust (Freeman MD, Kohles SS. Assessing specific causation of mesothelioma following exposure to chrysotile asbestos-containing brake dust. Int J Occup Environ Health. 2012 Oct-Dec;18(4):329-36.)

You have asked for my opinion regarding the diagnosis of Mr. Toy's disease, and whether his disease was caused by exposure to asbestos. I have reviewed medical records and pathology materials of Mr. Toy, as well as a work history containing asbestos exposure information provided by the Dean, Omar, Branham, Shirley law firm.

CLINICAL HISTORY:

Progress note (July 25, 2013) indicated evaluation of an abnormal chest x-ray with a history of asbestos related pleural disease, asbestosis and a history of smoking. Smoking history was reported as one pack per day for 30 years having quit in 1970. Prior work for the government and shipyards as a machinist reportedly resulted in significant asbestos exposure for about 14 years. An abnormal chest x-ray was evaluated. CT scan from May 2013 reportedly showed bilateral calcified pleural plaques and mild sub pleural fibrosis at the lung basis. Mr. Toy reportedly denied shortness of breath. Social history again indicated past occupational asbestos exposure. Physical examination identified bilateral crackles upon auscultation. The assessment indicated Mr. Toy had significant asbestos exposure during work and shipyards for about 14 years

with CT scan showing evidence of asbestos related pleural disease and the examination including CT scan confirming mild asbestosis.

Pulmonary consultation note (September 19, 2018) indicated a chief complaint of pleural effusion with a history of benign prostatic hypertrophy, chronic kidney disease, hypercholesterolemia, hypertension and asbestosis. Progressive shortness of breath was noted over the preceding two – four weeks. Prior CT scan reportedly showed bilateral calcified pleural plaques and a recent chest x-ray identified a moderate left pleural effusion and pleural plaques. The assessment indicated pleural effusion and pneumoconiosis due to asbestos.

Procedure note of thoracentesis (September 26, 2018) indicated obtaining 1760 mL of hemorrhagic pleural fluid.

Consultative report (September 27, 2018) indicated presentation with a left pleural effusion. CT scan identified bilateral pleural plaques consistent with the history of asbestos exposure. Social history indicated prior asbestos exposure while working for the government. A history of smoking one pack of cigarettes per day for 20 years, 40 years previous, was reported.

Operative report of video assisted thoracoscopy identified multiple areas of pleural plaque covering the entire pleura from the apex to the diaphragm within the left chest. Multiple biopsies were obtained.

Consultative report (October 4, 2018) indicated a diagnosis of “malignant mesothelioma”. A history of prior asbestos exposure was reported. Multiple pleural based nodules were reportedly biopsied and proven to be malignant mesothelioma. Malignant mesothelioma was deemed to be unresectable. Treatment options were discussed.

Discharge summary (October 4, 2018) indicated a primary diagnosis of “malignant mesothelioma”.

Office note (October 10, 2018) indicated presentation for discussion of treatment options regarding a new diagnosis of malignant pleural mesothelioma, epithelioid type.

Death certificate from the state of California listed the cause of death as “mesothelioma, etiology unknown”.

RADIOLOGY REPORTS:

Chest x-ray (September 15, 2018) identified right pleural calcification likely representing calcified pleural plaque.

CT scan (September 27, 2018) identified a small residual left pleural effusion and asbestos related pleural disease with probable parenchymal scarring in the right lung base.

PATHOLOGY REPORTS:

Report from Butler Memorial Hospital (CN 18 – 1509, September 27, 2018) diagnosed a left pleural fluid specimen as “atypical cells present”. Atypical cells reportedly sustained positive for 65/6, calretinin WT1 but negative for MOC 31, TTF1 and Napsin A.

Report from Butler Memorial Hospital (CN 18 – 1524, October 1, 2018) diagnosed a left pleural fluid specimen as “atypical cells present”.

Report from Butler Memorial Hospital (SU 18 – 16234, October 1, 2018) diagnosed multiple left pleura biopsies all as “malignant mesothelioma”. The comment reported tumor cells positive for calretinin, 65/6, WT1, D2 40, CD 141 and focal/week MOC 31 but negative for TTF1 and Napsin A. The histologic and staining patterns are all reported as supporting the diagnosis of malignant mesothelioma.

PATHOLOGY MATERIALS:

I received one paraffin block label “CN 18 – 1509”. I created one H&E stained glass slide. Microscopic examination demonstrated cellblock section of a fluid specimen showing blood elements along with clusters of large malignant epithelioid cells. Scattered malignant cells showed large regular, round and oval nuclei with multiple prominent nucleoli and voluminous eosinophilic cytoplasm. Tumor cells were arranged in small clusters. No gland, mucin or keratin formation was detected.

I received one paraffin block labeled "CN 18 – 1524". I created one H&E stained glass line. Microscopic examination demonstrated cellblock sections of a fluid specimen showing copious blood elements and innumerable clusters of large malignant epithelioid cells. Tumor cells were identical to those described above showing large pleomorphic nuclei with vesicular chromatin and abundant cytoplasm.

I received three paraffin blocks all labeled "SU 18 – 16234". I created one H&E stained glass slide from each block. Microscopic examination demonstrated sections of parietal pleura and chest wall soft tissue extensively involved by a malignant epithelioid neoplasm. Tumor cells were again identical to those seen in fluid specimen. Tumor cells were large with moderate to marked pleomorphism showing larger regular come around and oval nuclei with vesicular chromatin, mitotic figures and abundant eosinophilic cytoplasm. Tumor cells were arranged in nests and sheets with destructive invasion throughout pleura and into chest wall soft tissues. No gland, mucin or keratin formation was detected. No alveolar lung parenchyma was available for review.

Based on my review of records and tissues, my diagnosis is as follows:

Pleural fluid, left, thoracentesis (CN18 – 1509 & 1524):

- **Primary Pleural Malignant Mesothelioma, epithelioid type.**

Parietal pleura, left, biopsies (SU 18 – 16234):

- **Primary Pleural Malignant Mesothelioma, epithelioid type.**

ASBESTOS EXPOSURE HISTORY:

An asbestos exposure history letter provided by the Dean, Omar, Branham, Shirley law firm reports Mr. Toy was exposed to asbestos from approximately 1953 until 1980 while serving in the United States Army, as a Marine machinist at Hunters Point Naples Shipyard, and as a maintenance machinist at Treasure Island Naval Station. From approximately 1953 through 1959, Mr. Toy served as a wheel vehicle mechanic and then a motor sergeant. His job was to maintain the Army's fleet of vehicles during this time. The Army had approximately 50 2 ½ ton, ¾ ton, and ¼ ton vehicles. Mr. Toy performed brake, clutch, and gasket work on the vehicles, including sending of new clutches and

brakes and blowout of brake drums and clutch discs with compressed air during removal. He worked in a motor pool next to other mechanics. The motor pool had 50 bays. There was no ventilation. Spent eight hours a day during this time period in the motor pool working on and around others working on the Army's fleet of vehicles. He swept up dust from his work every day.

From approximately 1962 through 1973, Mr. Toy worked as a Marine machinist at Hunter's Point Naval Shipyard. As a result, Mr. Toy was exposed daily to asbestos containing gaskets and insulation associated with valves, pumps, turbines, generators, compressors and steam traps. His duties included the installation and removal of asbestos-containing gasket materials and the removal of asbestos-containing insulation materials. From approximately 1974 until 1980, Mr. Toy worked as a maintenance machinist at Treasure Island Naval Shipyard. He received daily exposure from asbestos gaskets and insulation associated with steam traps, valves, pumps and compressors. His duties included daily removal and replacement of asbestos-containing insulation and gaskets associated with valves, pumps, and compressors, and the installation and removal of brakes and clutches as a professional vehicle mechanic for approximately six months.

Mr. Toy was also exposed to asbestos-containing brakes, clutches and gaskets while performing work on his family vehicles from the early 1950s until 2016.

SUMMARY:

Based on my review of the medical records including pathology materials, I conclude that Mr. Toy had malignant mesothelioma of the pleura. The exposure history of Mr. Toy provided to me identifies a significant history of asbestos exposure. Mr. Toy developed malignant mesothelioma after an appropriate latency period following his first known exposures to asbestos.

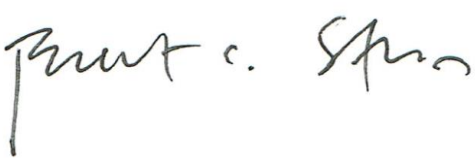
The occurrence of mesothelioma is associated with asbestos exposure, and mesothelioma is considered by many sources to be a signal tumor, meaning it signals prior asbestos exposure. Scientifically and medically speaking, it is not one asbestos fiber or one exposure, to the exclusion of other exposures that causes a person's

mesothelioma. Mesothelioma is caused by the totality of asbestos exposures, often called cumulative dose, that an individual is exposed to over his or her lifetime, taking into account an appropriate latency period. A mesothelioma occurs only after repeated exposures to asbestos over time, which in turn causes repeated damage to the mesothelial DNA and leads to mutations. It is well established that even brief or low exposures to asbestos can cause mesothelioma; however, it is also important to recognize that mesothelioma is a dose response disease. That means from an epidemiologic perspective, the greater the dose of asbestos, the greater the chance a given individual will develop mesothelioma, thus smaller exposures contribute smaller risks and larger exposures contribute larger risks. It is well established that exposure to both chrysotile and amphibole asbestos causes malignant mesothelioma. When I review an individual's exposure to asbestos and evaluate the causation of disease, as I have done in this case, I do not state that any contributor to the cumulative dose, no matter how small, is a significant factor to the development of mesothelioma. Rather, I review, evaluate, and consider the information available to me about an individual's identified exposures to asbestos, and only after that review will I consider causation and attribution of the asbestos exposures. My review is done in the context of additional opinions and foundation as stated in my affidavit of April 15, 2016, provided separately or attached to this report.

Based on the information available to me, that I have reviewed in this case and is described above, Mr. Toy had significant exposures to asbestos over his working lifetime. It is my opinion to a reasonable degree of medical certainty that Mr. Toy has a malignant mesothelioma that was caused by these identified and substantial exposures to asbestos.

I will supplement or amend this report as appropriate, if additional information becomes available.

Sincerely,



Brent C. Staggs, M.D.